JEFFRIES (J. A.)

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BY

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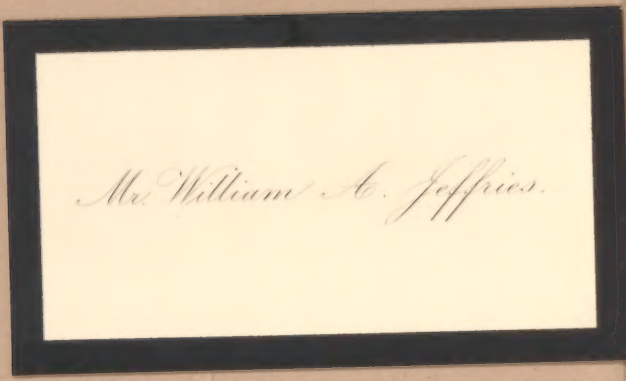

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EYE PARALYSES.*

BY JOHN AMORY JEFFRIES, M.D.

[This paper, which Dr. Jeffries had nearly finished at the time of his death, has been put in my hands to prepare for publication. Although I knew something of his intentions in regard to it, a study of the paper itself and of the mass of material which he had collected on the subject has convinced me of the difficulty of completing the task as he would have done it. With the exception of a few verbal changes, I have done little except to prepare a bibliography from the notes which Dr. Jeffries left, and to indicate in brackets the references to illustrative cases. Not having the original articles at hand and depending almost wholly upon these notes I fear that the classification of the cases, which I have tried to make, is not always correct, and that it might not agree with Dr. Jeffries' greater knowledge. I have added Bleuler's diagram, which Dr. Jeffries had copied, probably with the intention of inserting it. — P. C. K.]

PROBABLY every practitioner has at times been in doubt as to the nature of a case of eye-paralysis. The whole question is involved in practical difficulties. A patient complains of double vision; and it is evident, unless the case be one of monocular diplopia, that at least one of the twelve extrinsic muscles of the eyes is paretic. In theory, the changes in the relative position of the images in different parts of the visual field should lead to an accurate diagnosis; in practice, owing to the complex results produced when a number of muscles are affected, and to the stupidity of patients, the result is often far from satisfactory. Even when the eye distinctly lags in following an object, it may be difficult to determine the condition of the oblique muscles. When, as sometimes happens, the patient fixes with the paretic eye, there is danger that the trouble may be assigned to the wrong eye. The pare-

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tic eye is used for fixation when it has the best sight or when it is specially trained, as in the case of the right eye in surveyors. It is therefore desirable to make a systematic study of every case of eye-paralysis with all the methods at our command.

For this purpose it is necessary to have a pair of spectacles carrying plane glasses, one colorless, the other red, so that the color of the image may indicate to which eye it belongs. After assuring one's self that the diplopia is not of monocular origin, by having the patient look first with one eye and then with the other, the whole field should be tested with some bright object, as a candle-light, and the result noted in a chart-like manner. The relative position of the images, their parallelism or obliquity, the distance between them, and which is red, should be determined for at least three points directly in front of the patient, and for two tiers on each side representing half-way and full lateral vision. When, as is often the case, vision is impaired as well as the motions of the eye-ball, care must be taken lest monocular vision due to lack of vision in the peripheral portion of one retina be mistaken for proof of parallel vision. After this, near vision should be tested in the same way.

The double images had better be studied first, since they require close attention on the part of the patient and error is not easily recognized by the examiner.

It must be borne in mind that lack of double vision does not prove the absence of paralysis. One of the images may be disregarded, lost, or there may be but one, as in cases of conjugate paralysis, where there is constant parallelism of vision even though motion to one side is absolutely lost.

This is hardly the place to go into the details of the position of the images. They have been so fully worked out and figured in the various works on the eye and nervous system as to be familiar to all. Those

interested in the difficult subject of double paralysis of the superior obliques will find the subject elaborated in a recent article by Pflüger.[114] This paralysis is probably best recognized by the perimetic method.

Next the motions of the eyes should be observed when following some object, as a finger, a piece of paper, or the like. With practice very slight deviations from the normal can be recognized in this way. Not only the parallelism of vision, but the range of motion should be noted; also the position of the lid, and whether it follows the pupil in looking down or not. Very good motor charts can be made by Landolt's method in cases where the defect of motion is considerable. This consists simply in substituting a card with letters on it for the plain moving card of a perimeter, and after having instructed the patient to follow it as long as he can and still read the letters, slowly move the card from the centre to the periphery. The point where distinct vision ceases should be noted on any of the charts used in plotting out the field of vision. A muscular defect shows itself as a limitation in the part of the field towards which the weak muscle should have moved the eye. The method is based on the fact that the letters cannot be read unless their images fall on or very near the visual axis of the eye. Those who have not used this method, might think that it was entirely dependent on the promptness and accuracy of the patient's replies. This is not the case, as the reflections from the cornea render it very easy for the physician to recognize the faintest motion of the eye. Of course, this method fails entirely in those relatively rare cases where the paralysis is dependent upon binocular vision.

Several other factors remain to be determined which are more physiological in their nature; thus, it should be noted if there is any difference in the motion of the eye when its mate is covered. A quite marked or

even complete paralysis may vanish at once if the other eye be covered, while in others the paralysis is only apparent after the other eye is closed. Secondary deviation of the sound eye should be noted by placing a screen between the eye to be tested and the object gazed upon, and then removing it; if the deviation exists, the eye will be seen to fly back a few degrees as the object comes into view. It is due to the correlated muscles of the sound eye rotating the eye beyond the line of parallel vision, from sympathy in the effort required to fix the object with the paretic one. Hence, the direction of the motion is that in which the other eye is weak.

In making this test the screen should be held sufficiently near the eye to prevent fixation being performed by it and yet in such a way that the eye can be seen by the observer. The readjustment of vision is at times very quick, and the process may be finished before it can be noted.

False projection, if well developed, is also a factor of considerable value. This — the failure properly to locate objects in space — is held to be due to our judging of the relation of objects to ourselves not primarily by the position of their images on our retinæ, but by the state of construction of the extrinsic muscles of the eye. We first determine the direction of the objects falling in the line of distinct vision by the eye muscles and then locate the rest of the field by means of the relation of the peripheral images to the centre of the field. As a result of this, if a small object — say a pin stuck into a table — is fixed with the paretic eye, and the patient be instructed to pick it up quickly the hand will miss the object and go too far in the direction towards which the motion of the eye is weak. Of course, in making this test, the other eye should be closed, the motion made rapidly, and the pin placed so as to involve the use of the affected muscles. When

the paretic eye is closed, no false projection occurs, and the hand goes straight to the object. When both eyes are open, false projections in the opposite direction may result from overaction of the muscles of the healthy eye. When both eyes are affected, the last two tests often give most perplexing results.

Besides the above, the reaction of the pupil to light, near vision, and sensations from the skin should be noted as well as the action of the ciliary muscles. At the time of, or shortly after, the occurrence of a paralysis, the eye is apt to deviate in the opposite direction owing to the contraction of the antagonists, the same as in other parts of the body. But, in spite of this, the eye is often able to move in the direction of the paralyzed muscles as far as the mid-point.

The result of the examinations gives us a knowledge of the muscles paralyzed, the degree of paralysis, and the conditions under which it occurs. These facts have still to be correlated with what we know of the physiology of the eye, both normal and pathological, as well as with the results of clinical observation and the post-mortem examination before a full diagnosis can be made, and the physician be truly said to understand the case. A full diagnosis calls for a determination not only of the parts paralyzed, but also of the portions of the nervous system affected, and the nature of the affecting process. Fortunately for the patient, the second part of the diagnosis has little to do with treatment; only in surgical cases is an exact knowledge of the seat of disease called for by therapy.

The physiology of the muscles of the eye is by no means a simple problem; not only do we have to explain how the muscles bring the eyes into the proper position, but also to trace out the track through the nervous system by which this is effected. In the normal inactive state, where vision may be said to be in abeyance, the eyes rest in a mid-position and with

nearly parallel visual axes. I say nearly, because on several occasions I have thought that the eye did not correspond by a few degrees, the same as is common in the blind. Now, if an object to one side of the axis attract attention, the eyes are at once turned so that their axes are brought to bear upon it. This is done by the equal and synchronous action of the internal rectus of one eye and the external rectus of the other, and if the object is not on a level with the eyes, by the action of the muscles turning the eye up or down (superior rectus and inferior oblique or the reverse). This process, lateral conjugate deviation, has attracted much attention, and yet we do not know how it is brought about. Starting with the experiments of Mott and Schaefer [95 96 130] and others, we know that this motion is produced by stimulation of the middle portion of the frontal lobe just in front of the head area, the eye turning to the opposite side. Again, stimulation in the visual field in the occipital lobe causes the same motion, but in an opposite direction — that is, the eyes turn to the side stimulated. Lastly, if the seat in the occipital region where the eyes are turned to one side and that in the frontal region where the eyes are turned to the other are stimulated synchronously with the same current, the motion induced by the frontal area prevails over that of the occipital.

Over and above this, conjugate motion is liable to be induced by stimulation almost anywhere in the cortex. Such conjugate paralysis or deviation has been well recognized since the thesis of Prévost [118] and is one of the most constant early symptoms following almost any sudden gross lesion of the cerebrum. During the comatose period, if there be any, the eyes will be found turned as if looking to the sound side. When consciousness returns, the eyes may still continue in their former position, or assume the normal one. If still turned to one side, a true paralysis may exist, or

the position may simply be one of selection, the eyes being able to follow an object well to the opposite side. In either case the trouble usually vanishes in the course of a few hours or weeks, and the eyes return to a normal state. Since in pontine trouble the paralysis is crossed with that of the limbs, the symptom is at times of value in locating the lesion.

In a small proportion of the cases the deviation remains constant, as a permanent paralysis, indicating that the motor region or fibres have been actually injured and not simply disturbed in their functions. Efforts have been made to use this symptom for purposes of localization, but so far with poor success. Thus Wernicke [164] holds that such a paralysis coming on with a shock without loss of consciousness points to disease of the lower temporal region of the opposite side, and cites a few cases besides his own. They are, however, far from satisfactory.

There is a small number of cases where disease of the frontal convolutions has been accompanied by disturbance of paralysis of the eyes of such nature as to suggest some close connection with the lesion, but they are far too few and vague to justify any conclusions, though seeming to tally well with experiments on animals.

As we do not know whence the motions of the eyes spring, it goes without saying that we do not know the course of the fibres conveying the impulse to the nuclei at the base of the brain. They probably pass down in the anterior part of the internal capsule and thence, via the pyramidal tracts, to near the nuclei of the third nerve where they cross. Perlia [111] describes a set of fibres which, coming from the crus, turn and pass back along the râphe and then enter the nuclei from their free surface; these seem to be best explained as being the lower end of the cerebral fibres. The fibres to the sixth nuclei also probably cross some-

where in the same region, since in pontine trouble paralysis of the sixth nerve is always on the same side as the lesion. The sixth nucleus is placed under the eminentia teres in the angle formed by the knee of the facial nerve. Its root fibres pass down and out; the course of its cerebral fibres is not known.

Conjugate deviation in cases of pontine disease is of far more diagnostic value then when springing from disease higher up, and has, since Foville[82] first called attention to the subject, offered material for much discussion and many theories. In 1885 Bleuler[8] collected the cases and made a very material contribution to our knowledge of the subject; recently I have been at some pains to go over the literature and have been able nearly to double the number of cases with autopsy collected by Bleuler. Even a brief citation of these would swell this article far beyond all limits, nor in view of Bleuler's article is it necessary; but as they show several points of importance, a close summary is required. Owing to the internal recti being supplied by the third nerves, while the external recti receive their fibres from the sixth pair, lesions in the pons are liable to split up conjugate motion into its separate elements.

The following different states are found to exist:

(1) Complete paralysis of one external rectus and the crossed internal rectus, so that neither muscle is capable of any motion, the eyes looking forward or to the other side according to the state of the opposite pair of muscles. In these cases the eyes turn readily to the opposite side, up or down, but stop at the midline as if transfixed.

(2) Precisely the same state as in the first condition except that the internal rectus acts perfectly for near or convergent vision.

(3) When both eyes are open, the eye with the paralyzed internal rectus will not turn in for objects

on the other side of the nose, but will if the other eye be covered.

(4) The internal rectus will not act in conjugate, but will in near vision; the external rectus being normal.

(5) Though devoid of any signs of conjugate paralysis we must consider simple nuclear sixth nerve paralysis as an element of conjugate paralysis.

[Dr. Jeffries told me that he had omitted to speak fully of the cases in this first class, in which the conjugate paralysis was complete. I find in his notes references to eleven cases, which seem to belong here: those of Leyden,[54] Bernhardt,[5] Baller,[4] Broadbent,[18] Dennos,[35] Meyer,[54] Folville,[50] Garel,[80] Wernicke,[162] Mills,[88] and Webber.[161]]

In all these cases there were destructive lesions which, as far as can be judged from the report, involved either a considerable area in the pons above the nucleus of the sixth nerve or involved the pons immediately below or in front of the sixth nucleus on the paralyzed side.

In the second group of ten cases [Kahler and Pick,[97] Ferrol,[81] Vinantlis,[156] Mills,[88] Holstetter,[91] Grasset,[47] Poulin,[117] Blocq and Guinon,[9] Fanny,[37] Graux,[48]] eight showed a lesion just below or at the eminentia teres. It was in most cases small or at least did not affect much tissue in this region. In the other two, those of Mills[88] and Holstetter,[91] the lesion was a good-sized tumor in the upper quarter of the pons. The symptoms in the first case do not appear to have been in any way peculiar. In Holstetter's case there was simply a paresis of conjugate motion and no deviation to the opposite side.

I have only found five cases [Bleuler[8] (two), Spitzka,[138] Quinc,[117] Semelsohn,[127]] where it is clearly stated that the eye with the paralyzed internal rectus turned, as in conjugate motion, to the other side when the

other eye was covered. The question is how this comes about. To all appearance the suppression of the eye with the paralyzed external rectus restores power in the other, as if its weakness were due to sympathy and not to actual paralysis; but such an explanation not only fails to explain, but stands in marked contrast to peripheral paralysis of the sixth nerve where this action does not occur. There is nothing peculiar in the autopsies; in two there were tumors of moderate size below the sixth nucleus. In Bleuler's[8] two cases the tubercles, through diffuse, especially attacked the sixth nucleus. In Spitka's[144] the tumor, though extensive, apparently spared the floor of the ventricle along the râphe.

The only way out of the difficulty is to assume that the apparent conjugate motion in monocular vision is in reality a convergent motion substituted in its place. In this case we might expect the covered eye also to turn in and the pupils to contract. If either occurred, it would be fair to hold the point demonstrated. Unfortunately, in none of the cases has either been noted. But there are cases without autopsy, evidently of the same nature, in which this has been found to be the case. Also the obverse is known to occur in paralysis of convergence. If this view be correct, this group is really identical with the preceding.

There are three cases which apparently belong to the fourth group. Köchlin[71] reports the case of a boy three years old who after a month's sickness developed a decided deviation of the right eye to the right, and left facial paralysis. The tongue was projected straight; the pupils were equal; the limbs normal and the mind clear; pulse 112. After the paralysis had existed for a month, meningeal symptoms developed, and the child died. At the autopsy the only possible cause found for the paralysis was a tubercle adhering by its base to the floor of the fourth ventricle on the

left side at the level of the angle formed by cerebellar peduncles. The region of the third nerve nuclei was normal.

Hannius[97] reports the case of a man forty years old who had apoplexy. Two days later, up and down motion of the eyes was normal. There was absolute loss of power in the left external rectus, and the two internal recti; convergence and binocular vision could only be executed with difficulty (?); left conjugate motion lost; left eye in mid-position; right turned out forty degrees. Spinal myosis; marked dysarthria; right hemiplegia. Paralysis of right facial, the upper branch being least affected, and right hypoglossus. Head carried turned to the right, but can be turned anywhere. Mind clear, vision good, no nystagmus. By the fifth day the left eye had turned in a little, and the right was not turned so far out, only parallel to left. No other change. Death from pneumonia on the twelfth day. All the cranial nerves normal; softening in the left half of the pons beginning back of its anterior limit and most extensive in the middle. Part of the sixth root apparently implicated, but not the nucleus. Pyramidal tract entirely destroyed.

In the third case reported by Schutz[98] a woman forty-eight years old, after two weeks of weakness, was suddenly seized with paralysis and difficulty of speech, without loss of consciousness. On the fourth day there was diffuse headache, stertorous respiration, rapid pulse, and no œdema. She understood well, but spoke with difficulty. The head was freely moved, there was ptosis on the right, and the left eye did not turn in and down with the right; otherwise, motions of eyes normal. The pupils were small, the left angle of the mouth paretic, the tongue turned to the left. There was slight rigidity of all four limbs, and the right was paralyzed. At autopsy the vessels were found contorted, the ventricles enlarged, the epen-

dyma thickened, and a pair of small cysts the size of peas in the thalami. Also in the upper half of the pons an irregular hæmorrhage, larger than a hazelnut, in the region of the left seventh nucleus, which spared the lower parts on the left and part of the right half of the pons.

In the first case Gubler [30] has already given the interpretation accepted by me; the second case belongs, as far as loss of left conjugate motion is concerned, to the second group. It is only the internal rectus of the left eye which is here of interest. This acted in convergence and not in conjugate motion. The presumption is that the fibres of the third nerve system, affected in association with the system of the sixth nerve in conjugate motion, were here affected alone; but the autopsies are too vague to aid in plotting out the position of the fibres.

There is one case (Eichhorst [26]) of simple deviation of the eyes and no paralysis due to a lesion in the pons. A man forty-seven years old and a syphilitic, fell in coma after a few hours of moderate pain in the head. On return of consciousness there was right hemiplegia, motor aphasia, relaxation of right side of palate, right facial paralysis, dysphagia. The face and eyes were both turned to the right, but could be moved freely in any direction. There was also a tendency to lie on the right side. The pupils were much contracted and the light reflexes lost. Sensorium free. Death occurred on the eighth day. A clot was found in the anterior part of the basilar artery, and the pons was very soft in the anterior part of the left half. The softening extending nearly to the floor of the fourth ventricle. The cerebellar peduncles were normal. A figure of the basal surface of the pons shows that the softening involved the lower half of the left crus.

To my mind, this is not a case of conjugate paralysis but one of forced rotation to the right, a symptom

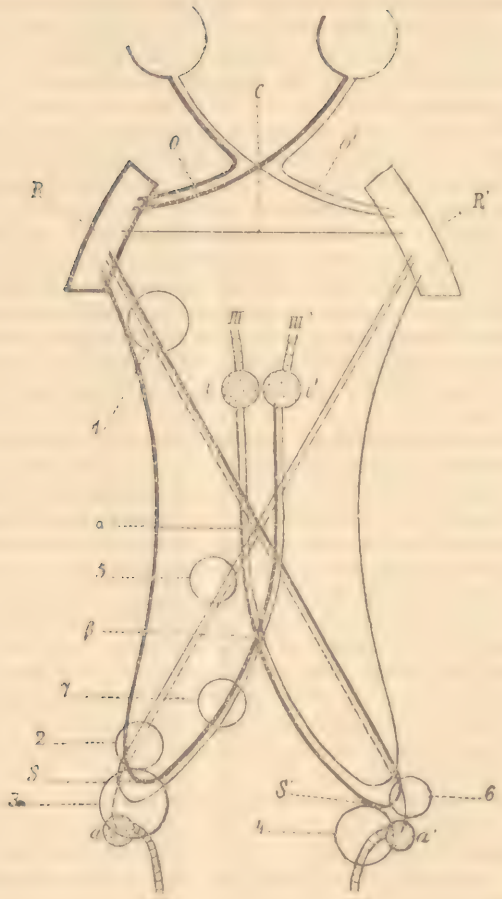
frequently associated with conjugate paralysis. There was no paralysis of the eyes. All the cranial nerves affected were on the same side as the hemiplegia, and the figure in spite of the text shows the lesions to be in the crus.

Since a central paralysis of the sixth nerve is an essential part of a conjugate paralysis, they are best considered in this connection. Bleuler* has classed a case reported by Grasset† among those of conjugate paralysis, which to my mind belongs to this group. The head, eyes, and body turned to the right, but the right eye could be turned to the left, while the left could not. Autopsy showed several small sarcomas, two of these being in the left half of the pons. This case seems to be in reality one of conjugate deviation of the eyes to the right and paralysis of the left sixth nerve.

My collection of simple paralyses of the sixth nerve dependent upon injury in the pons offers nothing peculiar in their character. There does not seem to be any point of distinction in action between the different parts of this system. In all, if not from the first, there have been before long other symptoms pointing to the pons. Some cases have begun as conjugate paralysis, and then become simple sixth paralysis; in others the obverse has been the case. In the first group the lesion has been a hæmorrhage at the eminentia teres. I have myself seen the two states alternate from time to time. Though there is no autopsy, a brief summary is given, as the case is of some interest.*

Considerable ingenuity has been spent upon the explanation of the mechanism of conjugate paralysis, and various theories evolved. Most of these involve either the existence of a special nucleus besides the sixth nucleus for conjugate motion of fibres arising from the

* No record of this case is given.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sixth nucleus and ending somewhere in the line for the internal rectus, either in the nucleus of the third nerve or the nerve itself. No one has pointed out the nucleus barring Gowers,[*] who suggests the olivary body, but as this is healthy in many of the cases it needs no consideration. The various fibre tracts have been pointed out by one or another, but not in such a way as to convince the doubter. None of these hypotheses stand in harmony with the facts.

Bleuler[†] has advanced a theory which was compatible with the facts up to his time of writing, and it holds good for the more recent cases. According to this, the fibres run direct from the cortex to their respective root nuclei, but there are two sets of fibres for the internal rectus, one for conjugate vision and one for near vision; the fibres run down as separate bundles below their nucleus close to the sixth nucleus, and then turn back and cross to their nucleus. There is no physiological objection to this theory, and it coincides well with the results of pathological investigations — indeed, it was built up from the best summary there is on the subject. It does demand the two sets of fibres, something not held to exist in any other part.

EXPLANATION OF FIGURE (from Bleuler).

O O′, Optic tracts.
R R′, Cortical centres.
a a′, Abducens nuclei.
i i′, Internal rectus nuclei.
S S′, Point where the internal rectus fibres bend.

1, Lesion producing Prévost's symptom.
2, [illegible] producing paralysis of one abducens and the opposite internal rectus for all movements.
4, Lesion producing paralysis of one abducens and weakness of the opposite internal rectus for all movements.
5, Paralysis of left abducens, absolute defect of right internal rectus for associated lateral movements, weakness of both internal recti for convergence.
6, Paralysis of abducens and inactivity of opposite internal rectus for lateral movements only.
7, Total paralysis of opposite internal rectus.

α, Double abducens paralysis.
β, Paralysis of convergence.

On the whole I believe it to be the best working theory we have. The only other way of looking at it seems to be that there is but one set of fibres, these going close to the sixth nucleus, but that the impulses descending from above in conjugate and near vision are different, and that the conjugate impulse is more easily interrupted than the near vision impulse; the nearest analogy being the different results obtained by the stimulation of a nerve as shown by Hooper and Bowditch, and the selection of the extensor muscles in most hemiplegias. Any such theory of course ignores the idea that each muscle is represented by certain nerve fibres, cells and areas, which transmit the force after the style of a complex system of bell cords, — an idea which seems in silence to be creeping into neurology, but does not tally well with complete recovery after resection of a nerve. One can hardly imagine that in these cases the cells in the cord establish their old relations with the muscles. If not, and the pull cord theory be correct, utter confusion should result when any motion is attempted.

Whatever the theory may be, the practical point remains that all cases of partial conjugate paralysis so far reported have depended upon lesions of the pons.

A few cases with autopsy have been reported in which conjugate paralysis of up or down, but not of lateral motion existed [Thomsen,[154] Nieden,[95] Hoppe,[59] Henoch,[54] Gee[42]]. Of these I have collected a few cases and have been unable to procure the records of several others. Some are cases of pure paralysis of up or down motion, others are complicated by other pareses but nevertheless this form of conjugate paralysis was the leading factor in the case. In all of these a lesion has been found at the level of the nuclei of the third pair, and in all but Thomsen's[154] case the disease has been central. As this case is of extreme interest it is given in detail:

A man, forty-eight years old, slowly developed the following state: Viscera normal, mind feeble; pupils small, right larger than left, a trace of the light reaction in the left, but none in the right. Lids drop in rest, but can be well opened. Both eyes moved to a normal extent to the right, to the left or down, but always with nystagmus. A marked paralysis on looking up, the eyes turning scarcely above the horizontal plane. Monocular vision the same, except that the right eye is less movable than the left. No changes in the state of the eyes to death one month later. The arms were weak and tremulous, the legs weak and slightly spastic with normal knee-jerks, but ankle clonus. The urine escaped at times, and the gait was unsteady and spastic. Autopsy revealed a moderate amount of chronic meningitis, thickening of the ependyma, a few sclerotic patches in the cord, and a partial degeneration of both the anterior and posterior roots. Also a gumma at the point of exit of the third nerves between the corpora mammillaria and the crura. In the left only a small portion of the crus and substantia nigra were affected, while on the right the growth involved the lower portion of the red nucleus, the median third of the substantia nigra, and less of the crus. The right third nerve was extensively degenerated, the left but slightly. The nuclei of the third nerve were normal.

Thomsen classes the case as one of peripheral paralysis, which to my mind is a mistake. Though the lesion was peripheral to the nuclei, it was still in the brain. The substantia nigra, and red nucleus can hardly be called peripheral structures. It seems clear that the lesion involved the nerve fibres destined for the superior and inferior recti.

The only other associated motion of the eyes it that of convergence in near vision. In this motion the eyes both bring their axes to bear on the subject, and ow-

ing to its nearness the axes converge. Contraction of the pupil and accommodation are normally associated with it. Commonly the object looked at in near vision is in front of the face, and hence the contraction of the internal recti are approximately symmetrical; but even in running the eye across a page this symmetry is broken. The extent to which lateral near vision can be practised appears to be very variable. In some cases as soon as the object is passed but a slight distance to either side of the nose, the pupil dilates, vision is poor, and the eyes become parallel, indicating the substitution of conjugate for convergent vision. In other cases the object can be carried so far to one side that one eye looks at the nose and the other is well turned out before the break comes. In all whom I have examined, even with the other eye covered, near vision does not occur when the object is far to one side.

A good many cases of paralysis of near vision have been reported, but unfortunately as yet there are no autopsies. [Cases by Barel,[2] Earles,[25] Parinaud,[108, 110] Peters,[112] Stölting,[150] Gräfe,[45] Pilz,[115] De Watteville,[21] and Binsler.[7]] Some of the cases are suggestive of an hysterical state, but the mass of them point to gross disease. A centre of convergence has been assumed to exist in the anterior part of the third nuclei, but as yet nothing definite has been shown. Hensen and Völckers'[55] experiments — which do not tally with others — at the most do not demonstrate a centre: irritation at a point of crossing of fibres would produce the same effect. Thus, if, as suggested above, the fibres governing convergence come down to near the sixth nucleus, ascend and cross to their nuclei, stimulation at this crossing would produce convergence. Whatever the mechanism this paralysis is just the obverse of lateral conjugate paralysis. With both eyes open, neither will turn in, but cover one and the other comes

in at once, while the covered turns out to a parallel position. The case reported by Bineler[7] may be taken as a typical example. A hard man, previously well, rose to an appointment and while blowing on the horn, suddenly lost sight of the music and had to stop playing. Careful examination showed that distant vision was good, but near vision poor and accompanied by crossed diplopia. When one eye was covered, the other turned in well for near vision, the covered eye turning out at the same time. The right pupil reacted to light, but did not to accommodation. Sense of cold less distinct on the right side than on the left. No other symptoms, no change in six months.

We have left to consider the great mass of eye paralyses, which simply affect one or more muscles and do not tend to select associated functions. In these cases we are deprived of a most valuable guide in localization, and are obliged to fall back on what other symptoms may be present, and a few isolated points of value. As is now well known, the nuclei of the fourth nerves lie just behind those of the third nerves, and practically form a unit with them. The nerves themselves, however, instead of passing down from the nuclei to the lower surface of the pons, go up and cross above the aqueduct before issuing from the brain. As a result, the crossing gives an occasion for double fourth nerve paralysis, without involvement of the other nerves; which the peripheral course of the nerves does not readily lend itself to. Though no cases of the isolated paralysis of the superior obliques have been reported, there are those of Christ's[8] and others in which this symptom has been used with good success.

There are few other factors of importance in the distribution of the paralyses. Matthes[9] and others have held that a peripheral paralysis of the nerve trunk was complete or practically evenly distributed among the various muscles supplied by the nerve, while a

paralysis due to disease in the base of the brain was commonly incomplete or irregularly distributed. Thus the intrinsic or extrinsic muscles of the eye alone might be affected or some of the muscles of the third and not the others; the reason being that while the nerve is compact and small and therefore not easily partly affected, the nuclei of the third nerves are strung along the floor of the aqueduct and the posterior part of the third ventricle in the form of several sub-nuclei. Various efforts have been made to determine the functions of these sub-nuclei, but so far little, if anything, beyond speculative results has been obtained. Anatomy shows that there is a median nucleus common to both third nerves, and that the posterior dorsal nucleus sends fibres across the râphe to the opposite third nerve. As a general axiom Mauthner's position is undoubtedly true; but, as the case of Thomsen already cited [154] and the following show, it is not absolute. Oppenheim[102] has reported two cases of brain tumor which appear probably to belong here. In the one there was paresis of the right third and sixth nerves, and of the left internal rectus from a tumor the size of an apple chiefly in the basal part of the right frontal lobe, but extending some to the left frontal lobe. In the other there was complete paralysis of right internal rectus with a tumor in the right temporal lobe which pressed upon the lower parts of the brain and third nerve. Meyer's [84] cases of multiple neuritis also bear on this point.

Another symptom of value is the variableness of the paralysis. Most true peripheral paralyses tend to run a definite course, and do not change rapidly, indeed from minute to minute, as is not uncommon in central cases. This is naturally explained by the nerves being little more than conductors, acting when isolated from the body, while the centres discharge and generate force. As a result of this it is not rare to see a slight

paresis grow during the course of an examination into a paralysis, and now paresis develop. From the same cause the paresis is apt to show itself at night or evening and not in the morning.

But the above characteristics are of use in only a small part of the cases: in the majority we must rely on the general symptoms and etiology for our diagnosis. There are a number of diseases which from their importance require consideration.

In tabes, as has long been known, there is frequently a history of transitory diplopia during the prodromal period. A patient sees double on and off, just for a moment, particularly at night. The time is fixed partly by the causes already referred to, and partly by the fact that lamps afford a close test of the eyes. When the eyes are tested no signs of paralysis are detected. In another group, usually more advanced, a positive paralysis exists, an external rectus is weak, convergence is poor, or they see double in the upper stories of the houses as they pass along the street. These paralyses may in their turn pass off in a few days, give place to others, or remain fixed. The origin of these fugacious paralyses is not known, but their significance when combined with previous syphilitic infection is gravely suggestive of tabes to follow. The more fixed paralyses are apparently due to a mixture of causes, the most common being a thickening of the ependyma and an extension of the process into the nuclei and fibre tracts; another is syphilitic infiltration about the roots of the nerves before they are combined into a compact bundle. Endarteritis and its results do not apparently play an important part in these early paralyses.

The latter and general paralyses of the eye occurring in tabes are usually classed as cases of ophthalmoplegia. These are characterized by their irregular distributions, extent and persistency.

Paralysis of the eye muscles is by no means rare in

cases of multiple sclerosis. Upthoff [157] found seventeen cases in a hundred. These cases are classed as follows: Two of double sixth paralysis, four of single sixth, three of third nerve, all partial; two of lateral conjugate, one of up conjugate, three of convergence, and two of ophthalmoplegia externa. It will be noticed that the paralyses are all limited, do not tend to include the whole of the third nerve, but only affect one or two muscles, as an associated motion. They tend to be more closely limited than in tabes.

Tumors involving the corpora quadrigemina, the pineal or pituitary gland are very apt to cause paralysis in the branches of the third, together with a peculiar form of ataxia and impairment of sight, but there is nothing in the paralysis itself to indicate the nature of the lesion, except perhaps in cases of paralysis of up-and down motion. The diagnosis of the nature of the lesion must be based on the general symptoms of tumor and the fact that other lesions are rare in this locality. In a summary of 29 cases Christ [17] found paralysis of the third nerve in 22, of the fourth in six, and of the sixth in nine cases.

The so-called cases of ophthalmoplegia externa require notice. In 1879 Hutchinson [64] called attention to a group of cases in which a progressive fairly symmetrical paralysis of the muscles of the eye balls formed a predominant symptom among a group of scattered bulbar paralyses and general cerebral symptoms. Since then the limits of this group have been extended so as to cover all general eye paralyses apparently of central origin and thus any value which may have attached to the name has been lost. To-day a diagnosis of ophthalmoplegia is about as significant as one of stomach ache. The twenty odd cases in which there are fairly good autopsy reports, some being monuments of labor, skill and knowledge, show a great variety of processes.

A few groups, however, can be separated out, with a fair degree of accuracy:

First, there are the cases of polio-encephalitis of Wernicke, represented by Gayet's case,[40] Wernicke's[107] three cases, and Thomsen's[108] two cases. In these cases there are but little paralysis except of the eyes, but in all mental disturbance, active delirium or somnolence, a staggering ataxic gait, tremor and the general signs of severe brain disease. At the autopsy a marked injection of the vessels, and numerous miliary hæmorrhages throughout the central gray matter of the third ventricle, aqueduct, and fourth ventricle have been the principal trouble. The process has also in some cases been diffused through the whole of this region. In others, more or less extensive degeneration of the nuclei has also been present.

In Hutchinson's case[109] the process is given as a nuclear degeneration, the same as in muscular atrophy, a pathology with which the symptoms well accorded. Some of the cases of diphtheritic paralysis also appear to partake of the same nature though peripheral trouble is present. These cases are slower than those of Wernicke's group, and lack the high degree of vascular change and acute symptoms, but seem to be allied to them by a certain amount of vascular change and the nuclear degeneration.

A second class is represented by the cases of Dubuys,[39] Eisenlohr,[29] and Bristowe,[17] in which careful microscopic examination gave negative results. In character they all differ: the first was a recurrent trouble, the second like a bulbar case, while in the third there were signs of Graves' disease and much suggesting hysteria.

The rest of the cases depend on all sorts of gross lesions, as Eitor's[41] with myelitis, Sutter's[61] with tumor, Buzzard's[16] with syphilitic endarteritis (?), while the changes occurring in company with tabes,

multiple sclerosis, and general paralysis make up the rest.

Dufour [24] has summarized the mass of reported cases of ophthalmoplegia exterior or nuclear palsies, from which the following figures are taken. Males 122, females 41. Before the fifteenth year, 23; in the next fifteen 35; and from thence to the sixty-ninth year 67 cases. Total, 125. Of 183 cases 31 had previous cerebro-spinal disease, 74 syphilis, diphtheria, diabetes, or the like, and 78 were in health.

Disease at the base of the skull often causes more or less paralysis of the eyes, as in tubercular meningitis and the so-called syphilitic meningitis. As a rule in the first any eye paralysis is of late date, and accompanied by affection of other nerves in a way suggesting a disease of the membranes, as well as by general constitutional symptoms. But this is no law, as the following case of Seitz [135] proves. A man of forty years went to bed well and woke up in the morning with a complete paralysis of the right third nerve. He later developed the full signs of tubercular meningitis from which he died. The autopsy showed nothing remarkable to explain the early eye paralysis. Syphilitic disease of the membranes is usually in reality a more or less diffuse gummatous or round cell growth, springing from the dura, and often lying between the dura and the skull. By creeping along the base of the skull and crowding the foramina such growths, though of small mass, can produce great mischief. It is equally true in these cases, as in others of syphilitic origin, that fever is conspicuous by its absence, while nocturnal pain and insomnia predominate. Unverricht's case [158] is a type of how a small amount of tumor can produce much mischief. In the other case the disease at the surface of the base is combined with central trouble depending on disease of the vessels or the neuroglia, as in the case carefully studied by Siemerling.[140]

Other tumors in the same region produce the same effects, but tend by pressure to cause paralyses of the body as well as optic neuritis. They also tend to a unilateral distribution, to picked paralysis of the fifth nerve, with neuralgia, as well as to involve the nerves in series as they run along the base of the skull.

There are still but few cases of recurrent paralysis of a third nerve with autopsy reported. (Richter,[129] Weiss,[130] Fiedler[35] two.) In all of these, in spite of the various theories advanced, some form of local disease of the nerve has been found as tubercle, fibroma, meningitis. These cases have not been true recurrent paralysis, since there has been a certain amount of paresis between the spells. They are cases of paresis of the third nerve with recurrent exacerbations. The spells are apt to be accompanied or preceded by pain, vomiting, and confusion.

Until lately there has been more or less feeling that the eyes were exempt in cases of multiple neuritis, a position exploded by the cases of Pal,[107] as well as by that of Meyer.[74] The last case is of special interest, as it partook of the character of an ophthalmoplegia. A man forty-two years, old suffering from chronic bronchitis and bronchiectasis developed a paralysis of all the muscles of the eye-balls and the levators of the lids, but with no paralysis of the pupils. Anæsthesia of the cornea, paræsthesia of the back and limbs, and dysphagia soon developed, shortly after which death occurred. A careful microscopic examination failed to show any disease of the brain, but did demonstrate an extensive multiple neuritis. The third, fourth, and sixth nerves were degenerated, while the facials, hyoglossals, glossopharyngeals and many spinal nerves were partially degenerated.

In disease of the orbit the paralysis may be general, according to accepted doctrine, or localized in certain

branches of the third nerve; but our knowledge of the subject is very meagre. The foramina themselves offer opportunity for trouble by the growth of exostoses, while fracture is not a rare cause of trouble. With so-called rheumatic paralyses of the eye muscles, so commonly referred to, I have had little or no experience. Of some fifty or sixty cases of which I have records, there is but one which can fairly be considered as belonging to this group if it exist. This case was seen but once, and was so classed from lack of any indications. It goes without saying that the probabilities of syphilis were great. The only autopsy made in the allied facial trouble (Minkowski [90]) revealed a general degeneration of the whole nerve, and no signs of rheumatism. Had other nerves been affected, the case would have been one of multiple neuritis.

There are a few other factors which require mention from the danger of their being overlooked, as congenital imperfections of motion due to paralysis, as in the cases of conjugate paralysis reported by Gräfe [46] and others, to imperfect insertion of the muscles or, to disease in the muscles and tissue of the orbit. Of the latter I have seen one case (following influenza?) in which there was much limitation of motion of the eyeballs, intense pain, deep tenderness, fever, pupils not affected, which recovered in ten days.

Ptosis, though not due to paralysis of a muscle of an eye-ball, is so closely connected with these as to require notice. The lid is raised by two muscles, the levator palpebræ supplied by the third nerve and unstriped fibres supplied by sympathetic fibres. Paralysis of the latter is not so very rare, judging from the slight dropping of the lid during a spell of hemicrania, and in tabetics. A slight congenital drooping of the upper lid is also by no means uncommon. This state is of importance chiefly from the possibility of its being mistaken for a true ptosis. The obverse con-

dition is seen in the failure of the lid to follow the pupil down and the wide palpebral fissure of Graves' disease.

True ptosis is a different affair and is a sign of importance as a danger signal rather than as a factor in diagnosis. It is commonly present in the early stages of hemiplegia from any cause, and has much the same significance as conjugate deviation of the eyes. Some writers have endeavored to localize a special centre for the lid in the temporal region, but have failed to make out a strong case. Barring the cerebral cases, ptosis occurs with paralysis of the third nerve, from almost any cause, and is often the first sign. A patient wakes up with slight drooping of one lid, and in the course of a few hours to months the other parts supplied by the third nerve are involved. Either the nerve to the lid is more exposed to disease, or owing to its position and constant work defects in the muscle are more easily recognized. However this may be, a picked paralysis of one muscle supplied by the third nerve is very rare and quite surely will be followed by others. The danger is that the physician may overlook or slight the paralysis and thus receive a disagreeable surprise.

No reference has been made to treatment, since, with so many different processes involved it would open up a wide field. The process causing the paralysis is the object to be aimed at, not the paralysis. My own feeling is that where the cause is not apparent, an eye paralysis is very apt to be of syphilitic origin, and as such to suggest antisyphilitic treatment. There is little pathological evidence at hand, but this view seemed in accord with the growing sentiment of those who have paid most attention to the subject. Unfortunately it does not follow that because an eye paralysis has a syphilitic basis, treatment will do any good.

To sum up:

(1) All cases of lateral conjugate paralysis are of central origin.

(2) When the paralysis is on the same side as other paralyses the lesion is on the opposite side of the brain. Such paralyses as a rule are transitory and follow almost any sudden lesion, and often only show themselves as a prevailing position of the eye, and not as a true paralysis or even paresis.

(3) When the paralysis is crossed with the paralyses below, the lesion is in the pons-medulla region.

The above three are equally true of spasms.

(4) A gradual development of conjugate paralysis clearly points to the region of the sixth nucleus of the same side as affected.

(5) Paralysis of up or down motions or both motions indicate disease in the region of the corpora quadrigemina, but may be due to disease in the third nerves proper, at the point of exit.

(6) Reasoning from analogy, paralysis of convergence points to disease in the central gray below the aqueduct, but as yet autopsies are lacking.

(7) Picked paralysis of parts of a third nerve strongly suggests central disease, but is not proof of it.

(8) A majority of the cases of eye paralysis occur in the syphilitic.

(9) A paralysis which changes rapidly, quickly showing fatigue, is probably central in origin.

(10) Transitory paralysis in the syphilitic is strongly suggestive of future tapes.

(11) An eye paralysis, however simple it may seem, is always a just cause for suspicion of trouble to come, and demands a prompt and thorough examination of the patient.

(12) There is no evidence that there is any form of connection between the sixth nucleus and the third, except in the cerebrum.

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